Two Cases of Coronary Artery Spasm Induced by Indocyanine Green

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SUMMARY

There is no evidence in the literature that coronary artery spasm is induced by indocyanine green (ICG). In the present report, we describe 2 cases who developed chest pain with transient ST elevation on electrocardiograms after intravenous administration of ICG.

Additional Indexing Words:
Coronary angiogram Electrocardiogram ST elevation Vasospastic angina

A reduction in coronary blood flow due to coronary artery vasospasm is now considered to be the main cause of Prinzmetal’s variant angina.1)-3) For the purpose of provoking vasospasm, many drugs and methods such as administration of ergonovine maleate or acetylcholine, exercise and hyperventilation have been developed.4)-9)

Indocyanine green (ICG) is usually used to evaluate liver function and cardiac output (dye-dilution method). We report here 2 cases who developed precordial pain with transient ST elevation on electrocardiograms following intravenous administration of ICG.

CASE REPORT

Case 1: A 56 year old farmer began to have precordial pain at midnight 4 weeks previously, and his pain increased gradually in severity and duration. He was admitted to Tottori University Hospital with severe chest pain on March 19, 1982, and was diagnosed as having had a myocardial infarction. He showed a good clinical course without any complications after admission. To evaluate cardiac performance, 20 days after admission he
underwent bicycle ergometer stress testing with hemodynamic measurements by the dye-dilution method using ICG. Before exercise, the blood pressure and electrocardiogram (ECG) were recorded, then hemodynamic measurements were performed by the dye-dilution method. One minute after intravenous administration of 10 mg ICG, severe precordial pain was experienced. At the same time, second degree atrioventricular block (Wenckebach type) and ST elevation in leads II, III and aVF were seen (Fig. 1-A). Chest pain was relieved and ECG changes improved within 2 min after immediate sublingual administration of nitroglycerin (Fig. 1-B). Serial measurements of creatinine phosphokinase (CPK), lactic dehydrogenase (LDH) and glutamic oxaloacetic transaminase (GOT) showed no abnormalities. On coronary angiograms performed 7 days later, as shown in Fig. 2, 10 mg ICG provoked a significant vasospasm in the right coronary artery associated with marked ST elevation in leads II, III and aVF. Intravenous administration of isosorbide dinitrate (5 mg) reversed these changes.

Case 2: A 66 year old businessman had been well until 1980 when he began to have anterior chest pressure. He was not treated in spite of medical advice. Since he had severe precordial pain early in the morning, he was
admitted to our hospital on January 18, 1982.

As ischemic heart disease was suspected, bicycle ergometer testing with hemodynamic measurement using the dye-dilution method was performed. One minute after injection of ICG, precordial pain appeared, and the ECG showed marked ST elevation in leads II, III and aV F (Fig. 3-A). Intravenous administration of isosorbide dinitrate (5 mg) relieved his symptoms and ECG abnormalities (Fig. 3-B).

DISCUSSION

Vasospasm is well recognized as the cause of Prinzmetal's variant angina.1)-3) Recently, the role of alpha-adrenergic receptors in the autonomic regulation of the coronary circulation has been well established, and the
activation of alpha receptors, which might be precipitated by paroxysmal
bursts of sympathetic hyperactivity caused by rapid eye movement sleep and
exertion early in the morning, could play an important role in the genesis of
vasospasm.\textsuperscript{10,11)\textsuperscript{10,11})

Since ECG changes during spontaneous attacks of vasospastic angina are
difficult to document, various provocative test methods such as ergonovine
maleate, cold pressure and exercise have been developed in order to detect
coronary vasoconstriction.\textsuperscript{4-9)\textsuperscript{4-9)}} Nevertheless, no method always provokes
vasospasm in patients with vasospastic angina, and some unknown factor,
which activates the sensitivity of coronary alpha-adrenergic receptors, might
contribute to the development of vasospasm.

In the present report, it is reasonable to assume that the cardiac symp-
toms experienced by the 2 patients after ICG injection were probably caused
by vasospasm, considering the ECG changes such as transient ST elevation.
In fact, coronary angiograms of Case 1 showed vasospasm in the right coronary
artery. Although the patients had no history of allergic disorders, one might suppose that ICG-induced reactions caused shock, which induced secondary ECG changes. However, there were no signs of shock such as depression of blood pressure and cyanosis. Moreover, both precordial pain and ECG changes (ST elevation) were improved immediately after administration of nitroglycerin and/or isosorbide dinitrate. Although it is difficult to explain why ICG induced ECG changes in the present cases, it is possible that ICG triggered coronary vasoconstriction under certain hypersensitive states of the coronary alpha-adrenergic receptors and/or sympathetic nervous system.

As far as we know, there are no reports of ICG provoking vasospasm, and therefore, the transient ST elevation induced by ICG is of interest because of its rarity. Since ICG might induce coronary vasospasm under certain conditions, ICG should be used carefully in patients with ischemic heart disease.

REFERENCES